Assessment Of Fetal Cardiac Functions In Pregnant Women Wıth Asthma

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Abstract

Aim:This study aims to investigate the effects of maternal asthma on fetal cardiac functions. Methods:The study was planned with 30 pregnant women who presented to a tertiary health center and were diagnosed with asthma and 60 healthy controls with similar gestational ages. The fetal echocardiographic assessment was assessed between 33 and 35 weeks of gestation with pulsed-wave Doppler (PW), M-mode, and tissue Doppler imaging (TDI). Fetal cardiac functions were compared between maternal asthma and control group. Cardiac functions were assessed according to the duration of maternal asthma diagnosis, as well. Results:Early diastolic function parameters, tricuspid E wave (p=0.001), and tricuspid E/A ratio (P=0.005) were significantly lower in the group with maternal asthma. TAPSE and MAPSE values were statistically lower in the study group than in the control group; p=0.010 and p=0.012, respectively. Parameters assessed with TDI (E', A', S', E/E', and MPI' of tricuspid valves) and global cardiac function parameters assessed with pulsed-wave Doppler like myocardial performance index (MPI) and left cardiac output (LCO) were similar between groups (p> 0.05). Although, MPI did not change between groups, and the isovolumetric relaxation time (IVRT) value was prolonged in maternal asthma cases (p=0.025). Conclusion:We found that maternal asthma disease causes alteration in fetal diastolic and early systolic cardiac functions, but the global fetal cardiac function does not change. Diastolic heart function values also varied with the duration of maternal asthma. Prospective studies are needed to compare fetal cardiac functions with additional patient groups according to disease severity and type of medical treatment.

Title

Assessment Of Fetal Cardiac Functions In Pregnant Women Wıth Asthma

Short Title

Fetal cardiac functions in maternal asthma

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Phone number: +905079320818**AbstractAim:** This study aims to investigate the effects of maternal asthma on fetal cardiac functions.**Methods:** The study was planned with 30 pregnant women who presented to a tertiary health center and were diagnosed with asthma and 60 healthy controls with similar gestational ages. The fetal echocardiographic assessment was assessed between 33 and 35 weeks of gestation with pulsed-wave Doppler (PW), M-mode, and tissue Doppler imaging (TDI). Fetal cardiac functions were compared between maternal asthma and the control group. Cardiac functions were assessed according to the duration of maternal asthma diagnosis, as well.**Results:** Early diastolic function parameters, tricuspid E wave (p=0.001), and tricuspid E/A ratio (P=0.005) were significantly lower in the group with maternal asthma. TAPSE and MAPSE values were statistically lower in the study group than in the control group; p=0.010 and p=0.012, respectively. Parameters assessed with TDI (E', A', S', E/E', and MPI' of tricuspid valves) and global cardiac function parameters assessed with pulsed-wave Doppler like myocardial performance index (MPI) and left cardiac output (LCO) were similar between groups (p$>$ 0.05). Although, MPI did not change between groups, and the isovolumetric relaxation time (IVRT) value was prolonged in maternal asthma cases (p=0.025).**Conclusion:** We found that maternal asthma disease causes alteration in fetal diastolic and early systolic cardiac functions, but the global fetal cardiac function does not change. Diastolic heart

function values also varied with the duration of maternal asthma. Prospective studies are needed to compare fetal cardiac functions with additional patient groups according to disease severity and type of medical treatment.**Keywords:** fetal echocardiography, asthma, pregnancy, fetal cardiac function.

Introduction

Asthma is a multifactorial, immune-mediated inflammatory disease. The pathophysiology of the disease is reversible lower airway constriction due to intermittent smooth muscle constriction in response to environmental allergens, infections, and irritants [1]. Asthma is a common chronic inflammatory disease in pregnancy and has become one of the most common public health problems worldwide. In general, asthma is thought to worsen with pregnancy in one-third of patients, improve in one-third, and have no significant change in the remaining patients. However, recent multiple case-control studies show that the percentage of asthma patients who worsen during pregnancy, 18.8%, is lower than previous data and is related to disease severity[2].

The diaphragm rises by 4-5 cm, and the subcostal angle increases by 50% with pregnancy. As a result, total lung volume and functional residual capacity decreased by 5% and 20%, respectively. Increased body weight also contributes to dyspnea during pregnancy. Progesterone is a stimulant of respiratory dynamics that can increase the sensitivity of the respiratory center to carbon dioxide, while estrogen increases the sensitivity of progesterone receptors in the respiratory system and is jointly involved in altering respiratory function [2]. Studies have shown that maternal asthma increases the risk of complications, including small for gestational age, low birth weight, congenital malformations, preterm birth, gestational hypertension and preeclampsia, diabetes, and perinatal mortality in fetuses and mothers. The cesarean section rate is higher in patients with severe or uncontrolled asthma during pregnancy than in the healthy pregnant population due to adverse perinatal outcomes [3].

Mediators of human basophil cells interact with environmental factors, allergens, and irritants. These mediators are associated with oxidative stress in the cord blood of newborns of mothers with atopic asthma[4]. Chronic fetal infections and hypoxia are examples of adverse conditions that impair cardiac myogenesis during complicated pregnancy and increase the lifetime risk of heart disease[5]. In one study, neutrophil-triggered inflammation of the placenta was found to lead to inadequate placental development and a lack of barrier function. As a result, maternally derived inflammatory monocytes from the placenta may migrate into the embryonic heart and alter the structure of cardiac tissue and the normal composition of resident cardiac macrophages [6].

The circulatory physiology of the fetus is different from that of the newborn. The right ventricle provides greater cardiac output from the second half of pregnancy until delivery [7]. Assessment of cardiac function by fetal echocardiography allows earlier detection of changes in cardiac morphology and may help predict perinatal and long-term cardiovascular outcomes. We hypothesized that chronic inflammation in pregnant women with asthma might affect fetal cardiac function. This study aimed to assess fetal cardiac function in pregnant women with asthma compared with a healthy group.

Methods Study Population This study was performed on 90 fetuses without cardiac abnormalities, including 30 fetuses of mothers with asthma and 60 fetuses of the uncomplicated healthy pregnant population. thirty pregnant women with asthma who visited the perinatology outpatient department of Ankara City Hospital in Ankara from May 2022 to October 2022 were included in the study. Fetal examinations were performed between 33 and 35 weeks of gestation. The study protocol was approved by the Medical Research Ethics Department of Ankara City Hospital and was conducted according to the guidelines of the Declaration of Helsinki. (E2-22-2392). Written informed consent was obtained from the study participants. Pregnant women diagnosed with preeclampsia, diabetes mellitus, and other chronic maternal and pregnancy-related diseases, multiple pregnancies, fetal growth restriction, fetal structural or chromosomal abnormalities, premature rupture of membranes, and taking medications other than antiasthmatic drugs were excluded from the study. Other exclusion criteria included maternal tobacco use and signs of fetal infection. Healthy pregnant women in a similar gestational week range were randomly selected as a control group. Maternal information, sociodemographic characteristics, and obstetric history were recorded. The year of asthma di-

agnosis and medication of the patients were recorded. Based on risk and exacerbation, asthma is classified as intermittent, mild persistent, moderate persistent, and severe persistent [8]. Our study included only patients receiving combination drugs such as inhaled corticosteroids and long-acting B2 agonists. Well-controlled asthma patients were included in the study according to the ACOG guideline[9]. All patients were asymptomatic at the time of the study. Asthma controls were intermittent throughout pregnancy, and no attack was observed.**Measurements**All ultrasound examinations were performed with the Voluson E8 ultrasound system (GE Medical System, Milwaukee, WI, USA) with a convex 2-5-MHz ultrasound transducer. All ultrasound examinations were performed by a perinatology fellow BLK and checked by FDÖ who is an associate professor and has almost ten years of experience in maternal-fetal medicine. First, the fetus was anatomically examined with 2DUS, the biophysical profile of all fetuses showed fetal well-being, and the estimated fetal weights were appropriate for gestational age. Standard fetoplacental Doppler indices were performed, including the determination of the pulsation index for the umbilical artery and middle cerebral artery. The fetal echocardiographic assessment was performed with two-dimensional (2D) imaging, M-mode imaging, pulsed-wave Doppler (PW), and tissue Doppler imaging (TDI). Doppler parameters were assessed in the absence of fetal motion. The angle of insonation was less than 20 degrees from the direction of blood flow, and measurements were made on at least three consecutive waveforms. These parameters were collected prospectively at 33 to 35 weeks gestation. Recorded perinatal outcomes included gestational age at delivery, body mass index (BMI), mode of delivery, birth weight, and APGAR scores. Mitral and tricuspid valve early diastole (E) and atrial contraction (A) were measured with PW Doppler by placing the cursor directly under the tricuspid and mitral valve leaflets in the apical four-chamber view. The E/A ratio was calculated [10]. Tissue Doppler was examined at the tricuspid annuli in the apical or basal four-chamber view in spectral Doppler mode. Peak velocities of the tricuspid valve in early diastole (E'), atrial contraction (A'), and systole (S') were measured with an insonation angle of less than 30 degrees without angular correction by placing the cursor on the lateral side of the tricuspid valve, adjusting the sample volume by 2-4 mm with TDI. Tricuspid velocity parameters were recorded in the same cardiac cycle[11]. The three most distinct waveforms and valve click with a fetal heart rate in the normal range (120-160 beats/min) were measured, and the values were averaged. E/E' indices were calculated for the tricuspid valve. After obtaining a clear image of the mitral and aortic valves in the five-chamber view of the heart, the sample volume was placed on the medial wall of the ascending aorta containing the aortic and mitral flows. Valvular clicks were used as cues for the calculation of each period. Myocardial performance index (MPI) was calculated from the waveform obtained (isovolumetric contraction time (IVCT) + isovolumetric relaxation time (IVRT)/ejection time (ET))[12].

Sample volumes were placed in the basal part of the right ventricular wall (tricuspid annulus). All velocities were measured during the same cardiac cycle, and no angular correction was applied. To calculate the right MPI' with TDI, the following time periods were examined: isovolumetric contraction time (IVCT'), ejection time (ET'), and isovolumetric relaxation time (IVRT'). MPI' was calculated as (IVCT' + IVRT') / ET' [11]. Measurements of mitral annular plane systolic excursion (MAPSE) and tricuspid annular plane systolic excursion (TAPSE) were performed on the free walls in the four-chamber view with apical or basal position of the fetal heart with M-mode imaging. The cursor was placed vertically at the atrioventricular junction, and measurements were performed. The maximum amplitude motion was considered as the magnitude of displacement between the end-systolic and end-diastolic regions and was measured in millimeters[13].

Statistical analysis IBM SPSS version 25 software (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, version 25.0. Armonk, NY: IBM Corp.) was used for statistical analyses. The sample size was calculated using G Power software (version 3.1 Franz Foul, Kiel University). It was determined that at least 30 participants had to be included to achieve a mean effect size of 0.50 and 95% power at a significance level of < 0.05 [14]. Control cases were planned as 60 participants as well. The Kolmogorov-Smirnov test was used whether the variables were normally distributed or not. Because the variables were normally distributed, descriptive analyses were reported as mean and standard deviation. A percentile calculation was performed for asthma duration. The top quartile (75th percentile) over twelve years of asthma duration was determined. Comparison of nonnormally distributed quantitative data (asthma duration) between two independent groups

was performed using the Mann-Whitney U test, and the data were expressed as the median (interquartile range). Student's t-test was used to compare the measured values of two independent groups, and Pearson's test was used for correlation coefficients. P value below 0.05 was accepted as statistically significant.

Results

The clinical characteristics and perinatal outcomes of the study groups are shown in Table 1. Gestational age (p=0.104) and BMI (p=0.145) were similar between groups. Maternal age (p=0.042), gravidity (p=0.010), and parity (p=0.017) were higher in the maternal asthma group than in the control group. APGAR scores at 1st and 5th minutes, birth weight, and gestational age at birth did not differ between groups.

Table 1. Clinical features and perinatal outcomes between the groups

	Asthma (N:30) Mean ± SD	Control (N:60) Mean ± SD	P value [a]
Gestational age at examination	34.06 ± 0.90	34.35 ± 0.76	0.104
Maternal age	29.23 ± 4.84	27.13 ± 4.38	0.042*
Gravida	2.70 ± 1.95	1.85 ± 1.13	0.010*
Parity	1.23 ± 1.05	0.65 ± 0.39	0.017*
BMI	25.84 ± 2.67	25.06 ± 2.17	0.145
Gestational age at birth	38.50 ± 1.04	37.96 ± 1.60	0.097
Birth weight	3257 ± 524	3123 ± 367	0.161
APGAR score 1 min	7.23 ± 0.72	7.35 ± 0.73	0.477
APGAR score 5 min	8.63 ± 0.66	8.61 ± 0.64	0.909
[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the p<0.05 level.	[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the p<0.05 level.	[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the p<0.05 level.	[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the p<0.05 level.

A comparison of the fetal Doppler indices and cardiac function measurements of maternal asthma and control groups is shown in Table 2. Doppler indices did not differ between groups. The Tricuspid E wave velocity and tricuspid E/A ratio measured by pulse wave were statistically different between groups. (p=0.001) and (p=0.005), respectively. These parameters were lower in the case group. Left ventricular IVRT was statistically different between groups (p=0.025) and was longer in the asthma group, 45.06±8.47 ms (mean±SD), than in the control group, 40.98±7.76 ms (mean±SD). When the M-mode technique was used, the mean values of the TAPSE (p=0.010) and MAPSE (p=0.012) measurements were statistically different between the groups. The mean value of TAPSE was 8.17±1.32 mm and the mean value of MAPSE was 6.97±1.39 mm in the case group, and these values were significantly lower in the asthma group than in the control group.

Table 2. Comparison of Doppler indices and fetal cardiac function measurements between the groups.

		Asthma (N:30) Mean ± SD	Control (N:60) Mean ± SD	P value[a]
Doppler Indices	Umb. A S/D	2.60 ± 0.40	2.46 ± 0.33	0.161
	Umb. A PI	0.97 ± 0.18	0.89 ± 0.18	0.083
	MCA PI	1.80 ± 0.36	1.64 ± 0.45	0.104

Cardiac Function	PW Doppler	Tricuspid E (cm/sec)	37.30 ± 5.52	40.73 ± 4.06	0.001*
		Tricuspid A (cm/sec)	58.56 ± 4.55	58.92 ± 5.46	0.758
		Tricuspid E/A ratio	0.63 ± 0.09	0.69 ± 0.07	0.005*
		Mitral E (cm/sec)	34.81 ± 6.27	33.84 ± 7.41	0.540
		Mitral A (cm/sec)	47.52 ± 7.98	49.97 ± 10.19	0.253
		Mitral E/A ratio	0.73 ± 0.09	0.68 ± 0.13	0.091
		LV IVCT (ms)	36.30 ± 6.65	34.13 ± 6.00	0.123
		LV ET (ms)	164.13 ± 20.95	160.41 ± 16.25	0.357
		LV IVRT (ms)	45.06 ± 8.47	40.98 ± 7.76	0.025*
		LV MPI	0.50 ± 0.09	0.47 ± 0.08	0.093
		LCO	340 ± 109	378 ± 104	0.111
		Tricuspid E/E' ratio	5.04 ± 1.92	5.00 ± 0.90	0.901
	TDI	Tricuspid E' (cm/sec)	8.07 ± 2.02	8.33 ± 1.35	0.465
		Tricuspid A' (cm/sec)	13.31 ± 3.11	13.27 ± 1.98	0.942
		Tricuspid E'/A' ratio	0.61 ± 0.09	0.63 ± 0.08	0.257
		Tricuspid S' (cm/sec)	11.53 ± 7.82	9.77 ± 2.20	0.109
		RV IVCT' (ms)	41.46 ± 9.26	40.60 ± 9.95	0.694
		RV ET' (ms)	159.33 ± 17.10	155.30 ± 17.44	0.302
		RV IVRT' (ms)	47.23 ± 12.13	44.50 ± 8.40	0.217
		RV MPI'	0.55 ± 0.11	0.54 ± 0.07	0.429
	M-Mode	TAPSE (mm)	8.17 ± 1.32	8.86 ± 1.09	0.010*
		MAPSE (mm)	6.97 ± 1.39	7.64 ± 1.03	0.012*
[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the $p<0.05$ level.	[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the $p<0.05$ level.	[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the $p<0.05$ level.	[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the $p<0.05$ level.	[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the $p<0.05$ level.	[a] Independent Sample-t test was used to compare the values of two independent groups. * values show statistical significance at the $p<0.05$ level.

A comparison of fetal cardiac functions as a function of asthma duration is shown in Table 3. We found that 12 years or more maternal asthma duration represented the top quartile of asthma duration. The asthma duration of 8 pregnant women with asthma was twelve years or more. The parameters of diastolic

dysfunction such as mitral E wave velocity (p=0.031), mitral A wave velocity (p=0.013), tricuspid E wave velocity (p=0.034), and tricuspid A wave velocity (p=0.020) were significantly different between the pregnant women with asthma duration of 12 years and more compared to pregnant women with asthma of less than 12 years.

Table 3. Comparison of fetal cardiac functions according to asthma duration

Asthma		Duration <12 years N:22	Duration [?]12 years N:8	P value[b]
Global Function	LV MPI	0.51 (0.47-0.56)	0.46 (0.39-0.57)	0.372
	RV MPI'	0.58 (0.51-0.66)	0.51 (0.48-0.57)	0.100
	LCO	320 (251-389)	341 (297-370)	0.851
Systolic Function	LV IVCT (ms)	38 (33-40)	34 (28-41)	0.383
	LV ET (ms)	165 (153-177)	164 (159-168)	0.622
	RV IVCT' (ms)	40 (33-47)	41 (37-47)	0.759
	RV ET' (ms)	160 (142-173)	165 (154-175)	0.302
	Tricuspid S' (cm/sec)	8.67 (6.69-10.04)	17.20 (7.91-19.78)	0.083
	TAPSE (mm)	7.73 (7.03-9.48)	8.15 (7.35-9.15)	0.870
	MAPSE (mm)	6.81 (6.20-7.45)	6.69 (5.85-8.62)	0.907
Diastolic Function	LV IVRT (ms)	45 (40-50)	36 (34-49)	0.063
	RV IVRT'	50 (42-58)	43 (40-47)	0.260
	Mitral E (cm/sec)	32.1 (28.9-38.5)	41.3(35.1-43.9)	0.031*
	Mitral A (cm/sec)	45.4 (40.0-48.1)	53.0 (48.5-61.7)	0.013*
	Mitral E/A ratio	0.75 (0.68-0.81)	0.71 (0.65-0.76)	0.399
	Tricuspid E (cm/sec)	36.8 (35.0-43.6)	35.3 (32.5-36.0)	0.034*
	Tricuspid A (cm/sec)	60.8 (56.0-62.9)	56.7 (51.9-58.5)	0.020*
	Tricuspid E/A ratio	0.65 (0.57-0.72)	0.63 (0.56-0.69)	0.606
	Tricuspid E' (cm/sec)	8.1 (6.6-9.1)	9.2 (7.1-10.0)	0.223
	Tricuspid A' (cm/sec)	11.7 (10.3-14.9)	15.3 (12.7-16.9)	0.087
	Tricuspid E'/A' ratio	0.64 (0.56-0.71)	0.58 (0.51-0.65)	0.214
	Tricuspid E/E' ratio	5.05 (4.01-6.00)	3.73 (3.10-5.17)	0.159
[b]Mann Whitney U test was used to compare the measurement values of two independent groups. Values were presented Median (IQR). * values show statistical significance at the p<0.05 level.	[b]Mann Whitney U test was used to compare the measurement values of two independent groups. Values were presented Median (IQR). * values show statistical significance at the p<0.05 level.	[b]Mann Whitney U test was used to compare the measurement values of two independent groups. Values were presented Median (IQR). * values show statistical significance at the p<0.05 level.	[b]Mann Whitney U test was used to compare the measurement values of two independent groups. Values were presented Median (IQR). * values show statistical significance at the p<0.05 level.	[b]Mann Whitney U test was used to compare the measurement values of two independent groups. Values were presented Median (IQR). * values show statistical significance at the p<0.05 level.

The correlation between the duration of maternal asthma and fetal cardiac functions is shown in Table 4. A positive correlation was found between mitral E wave velocity, A wave velocity, and duration of maternal asthma. A negative correlation was found for the tricuspid A wave velocity with maternal asthma duration.

Table 4. Correlation of maternal asthma duration and fetal cardiac functions

Asthma duration	
R value[c]	
P value[c]	

[c] Pearson Correlation test was used to determine the correlation between maternal asthma duration and fetal cardiac functi

Discussion

In our study, we aimed to investigate whether or not the inflammatory process associated with maternal asthma affects fetal cardiac functions. Fetal cardiac function measurements from pregnant women with asthma in the asymptomatic period of the disease and with uncomplicated pregnancies were compared with those from healthy pregnancies. Fetal right and left ventricular functions were studied using different ultrasound techniques such as pulse wave Doppler, spectral TDI, and M-mode. Using the pulse wave Doppler technique, we found that the tricuspid E wave velocity and tricuspid E/A ratio were lower in the fetuses of mothers with asthma compared with the control group. This finding showed us that an early diastolic change began in the fetuses of pregnant women with asthma. While MPI and LCO parameters, which are indicators of global cardiac function, did not change, it was observed that the left ventricular IVRT value, which is one of the MPI parameters, was prolonged in the asthma group, which is another marker of diastolic change in the heart. In the study performed with the M-mode technique, we found significantly lower MAPSE and TAPSE values in fetuses of pregnant women with asthma. A significant difference was found between an asthma diagnosis duration of 12 years or more and a duration of less than 12 years in both atrioventricular valves E and A wave velocities. We found that the duration of maternal asthma was negatively correlated with the tricuspid A wave velocity and positively correlated with the mitral E wave velocity and A wave velocity. In adult cardiac dysfunction cases, diastolic functions are affected in the early phase and systolic functions in the later phase. The E/A ratio reflects the ventricular inflow pattern, and this ratio is used to assess diastolic function or ventricular relaxation. Parameters such as annular displacement or velocities, which indicate longitudinal function, are typically impaired in the very early stages of fetal cardiac dysfunction compared with radial function parameters such as ejection fraction and cardiac output. Similarly, diastolic parameters such as IVRT are the first to be altered to reflect impaired relaxation[15]. The E/A ratio partly reflects cardiac preload, whereas the outflow tract reflects cardiac afterload and is often considered together. MPI is an early and reliable marker that indicates global myocardial function [16] and provides data on different periods during the systolic phase of the cardiac cycle. IVRT is the most important parameter of MPI affected in complicated pregnancies. In general, impaired ventricular dysfunction is associated with higher MPI values, which is due to the prolongation of IVRT. Increased IVRT is associated with decreased ET. IVCT is the most stable parameter of MPI [10]. Reduced E/A ratios and increased MPI values in IUGR fetuses have been demonstrated in the literature[17-19]. Interestingly, some studies found higher left ventricular MPI values and higher E/A ratios in fetuses with PPROM with microbial invasion of the amniotic cavity and with signs of fetal inflammatory response syndrome [20, 21]. In another study investigating fetal heart functions in maternal familial Mediterranean fever (FMF) associated with chronic inflammation, it was found that fetal diastolic functions might be altered in fetuses of mothers with FMF. As the duration of maternal FMF increases, systolic functions may also change [14]. In our study, we found that MPI and LCO parameters did not change, but E wave and E/A ratio values were lower in the asthma group. We also found a prolongation of IVRT in the asthma group. TDI is a method of estimating early preclinical cardiac dysfunction by measuring the velocity of myocardial movement. Comas et al. suggested that TDI may be a more sensitive method than conventional echocardiographic techniques for assessing fetal cardiac dysfunction[11]. TDI also allows measurement of MPI similar to pulsed Doppler. The E/E' ratio, the ratio of early tricuspid annular velocity (E') to early peak filling velocity of the tricuspid valve (E), is one of the

echocardiographic parameters of diastolic function in the prenatal period [7]. Some studies have investigated fetal cardiac functional changes in fetuses with IUGR using the s-TDI technique [18, 22]. Our study did not find any difference between the groups in the parameters examined by the TDI method.

The M-mode method can be used to assess the longitudinal motion of the heart. This approach is best suited for the examination of the right ventricle. Longitudinal cardiac function, which is driven by longitudinal myocardial fibers, is considered the first function to be affected in the presence of hypoxia. In a study, it was found that all annular parameters indicative of subclinical cardiac dysfunction decreased significantly in the IUGR group. In this study, it was found that M-mode examination showed the same performance as a classifier compared with TDI examination, especially in the tricuspid annulus[23]. In our study, we found that MAPSE and TAPSE levels were decreased in the maternal asthma group compared with the control group. However, asthma duration did not affect MAPSE and TAPSE scores.

Limitations

One of the major limitations of our study is that the sample group is small and it is a single-center study. In addition, inflammatory markers in maternal blood and cord blood were not studied. The results were not confirmed by echocardiography at postnatal follow-up of the newborns, which is another limitation of our study. There are insufficient data in the literature on the fetal cardiac effects of inhaled corticosteroid use. No subgroup analysis was performed according to the severity of maternal asthma and the type of medical treatment.**Conclusion**To our knowledge, this is the first study to examine fetal cardiac function in pregnant women with asthma. We found that maternal asthma affects fetal diastolic and early systolic cardiac functions, but global fetal cardiac functions do not change. Diastolic heart functions may be affected by the duration of maternal asthma. We believe that fetal echocardiographic assessment plays an important role in maternal systemic and inflammatory diseases. Further studies are needed to compare fetal cardiac function parameters in relation to disease severity and type of medical treatment with additional patient groups.

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Conflict of Interest

The authors declare that they have no conflict of interest.

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